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BLOOD CHANGES IN CONVULSIONS, ESPECIALLY IN EPILEPSY[1]

O. WUTH

From the Laboratory of Internal Medicine, Phipps Psychiatric Clinic, The Johns Hopkins Hospital

Convulsions are such a clearly defined and well-marked manifestation that nearly all research work dealing with metabolism in epilepsy has started from the convulsion. Naturally, an upheaval such as a convulsion produces marked changes in the blood. The methods developed mainly by Folin and his associates could not fail to facilitate and stimulate the examination of these changes, but, notwithstanding the amount of work done in this line, we still lack a

TABLE I

Convulsions

BEFORE	DURING	AFTER
Leucocytosis (Riebes)	Leucocytosis (Goedicke)	Leucocytosis (Itten, Riebes, Schultz)
Leucopenia (Hartman, Di Gaspero)	Lymphopenia (Di Gaspero)	Lymphocytosis (Riebes)
Lymphocytosis (Schultz, Itten, Zimmermann, Gorrieri)	Lymphocytosis (Zimmermann, Schultz, Rohde)	Eosinophilia (Gorrieri, Di Gaspero, Schultz)
Eosinophilia (Gorrieri)	Eosinophilia (Zimmermann, Schultz)	

clear picture of the blood-changes. The results of the various authors differ widely. The divergences of the previous results regarding cell-counts and differential counts are illustrated in Table I.

Regarding the changes in blood-chemistry I prefer to limit myself to a discussion of only those constituents which I have examined myself. Krainsky, Rohde, and Allers observed an increase of non-protein nitrogen in the blood after convulsions. De Crinis observed

[1] Received for publication February 18, 1926.

variations of the serum protein content; such variations were also found by Meyer, Bruehl, and Jacobi. Frisch found in some cases an increase of non-protein nitrogen before the convulsions.

All these authors, with the exception of Frisch, have formulated theories as to the etiology of the disease epilepsy on the basis of their findings. Thus Krainsky thought amidocarbonic salts were the cause of epilepsy, whereas Meyer and Bruehl look for the cause of epilepsy in endocrine disturbances and de Crinis sees a parallelism between epilepsy and anaphylactic disorders.

It seemed essential to me before drawing definite conclusions and forming final theories on inconclusive evidence to try to establish some definite facts. Utilizing my former investigations, I decided to increase my material in order to obtain more conclusive results. The questions I tried to answer were: (1) Whether any regularity of the blood-changes during convulsions could be found. (2) Whether the changes were always uniform in the same or in different individuals. (3) Whether the changes were specific for "genuine epilepsy." (4) Whether there was any relation between the observed changes and the severity of the attack. (5) Whether any evidence could be procured as to the nature of the changes.

The examinations have been carried out on five cases of genuine epilepsy, one case of traumatic epilepsy, one case of general paresis, two cases of brain lues, and one case of cardio-vascular-renal disease. These patients have been examined repeatedly in order to obtain a curve of the changes. In sixteen other cases only one blood sample has been examined immediately after convulsions, namely, in eleven cases of genuine epilepsy, three cases of general paresis, one case of cerebral lues, and one case of arteriosclerosis. I should like to remark that the time which is allowed to elapse between the convulsion and the drawing of the blood-sample is an important factor since the time needed for the compensation of the disturbances differs widely from case to case, and even in the same individual in different convulsions. Neglect of this factor probably explains to some extent the discrepancies of the previous results.

The examinations made were: cell count, differential count, serum protein content, non-protein nitrogen, uric acid, creatinine. The methods employed were: for the cell count, the Zeiss-Thoma pipettes

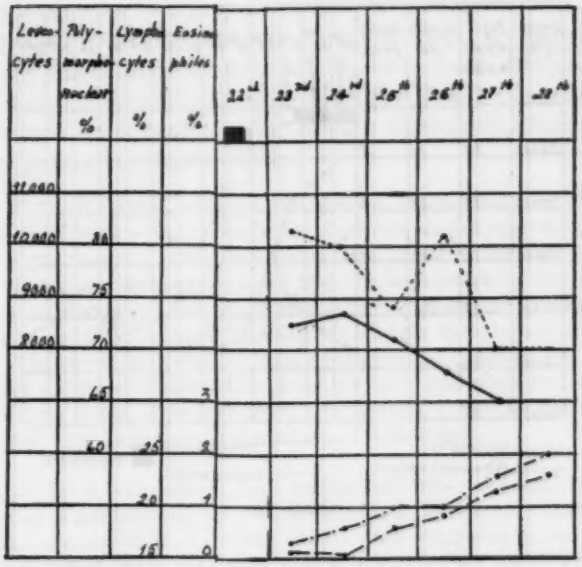

CHART 1a

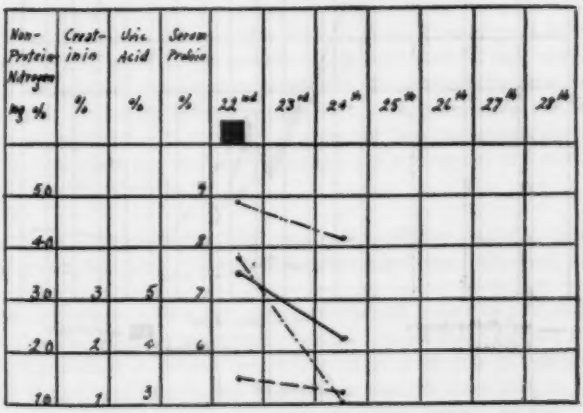

CHART 1b

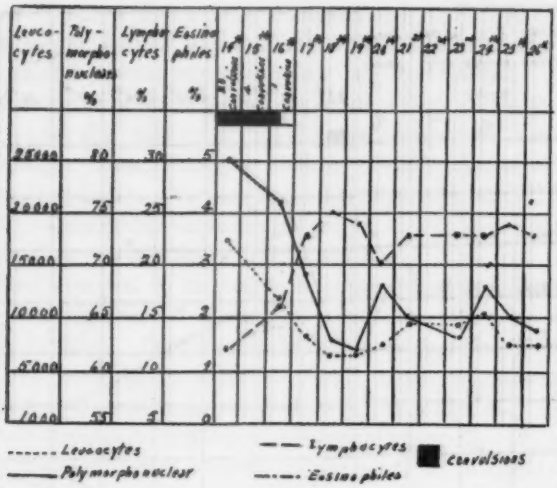

CHART 2a

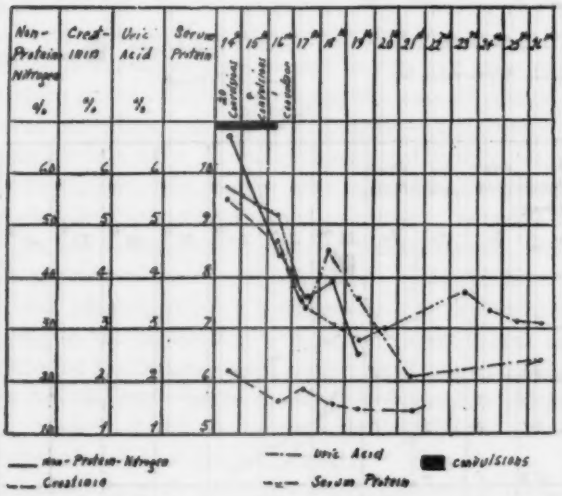

CHART 2b

and chamber; for the protein content of serum, the Zeiss dipping refractometer;[2] for coagulation, uranyl acetate; for blood sugar, Neubauer's hydroxylamine titration; for non-protein nitrogen, Pregl's micro-method; for uric acid and creatinine, Folin's methods.

The cases were studied at the Munich Psychiatric Clinic and the Henry Phipps Psychiatric Clinic.

Case 1. A. R., female, suffering from genuine epilepsy had four severe convulsions on April 22. The curves of the blood cells showed that the leucocytes fall after the cessation of the convulsions from 10,300 to the normal level of 8,000. The curve furthermore shows that the leucocytosis in this case consisted in an increase of the polynuclear cells. Lymphocytes and eosinophile cells are low at the time of the convulsions and later rise to the normal in a broken curve. The curves for NPN, uric acid, creatinine, and serum protein are very significant and characteristic in this case; high values immediately after the convulsions drop to the individual normal level within a variable but relatively short time. This is the most frequent type I have found.

Case 2. D., a man suffering from cerebral lues with occasional convulsions. On the day of admission he had twenty convulsions, the next day four, the day after that one convulsion. He was treated with venipuncture and amylene hydrate. The leucocytes on the day during which he had twenty convulsions were as high as 17,300. The curve shows that the white cells drop after the convulsions have stopped and, after some fluctuations, remain again within normal limits. The increase occurs in the polynuclear cells, as is demonstrated by the close comparison of the two curves. The lymphocytes show low values and do not change much; eosinophiles are totally absent in the period after the convulsions; later they rise rapidly, fluctuate, and then remain normal. The NPN was as high as 67.55 mg. per 100 cc., the uric acid 5.5 mg., creatinine 2.16 mg.; all these blood constituents dropped to the normal after the convulsions had ceased. The serum protein content during the convulsions was far beyond the normal limit with 9.77 per cent. In general, this case shows the same type of blood changes as the first case, although the etiology of the convulsions is evidently totally different.

Case 3. A man, suffering from traumatic epilepsy due to a lesion of the cerebral cortex, had several convulsions before admission. On Sept. 23 and on

[2] I am well aware of the fact that refractometer values in pathological cases do not give the exact serum protein content, but, all other authors having employed this method, I decided to use it in order to be able to compare results. Further, Berger and Petschacher have shown that, in cases such as those with which we are dealing, the method gives sufficiently correct results.

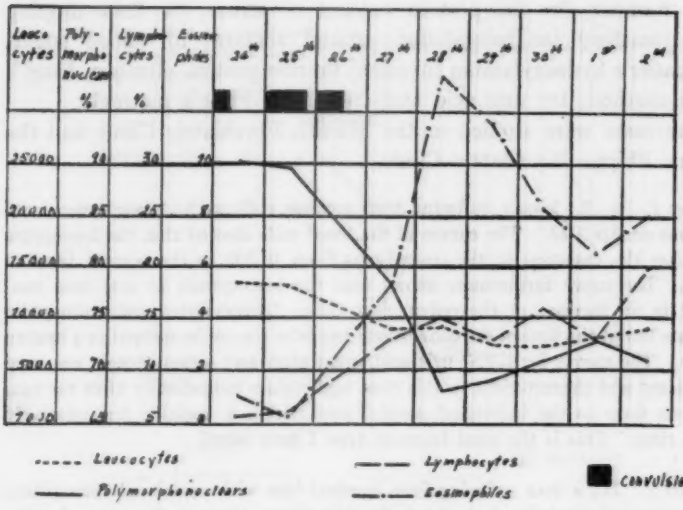

CHART 3a

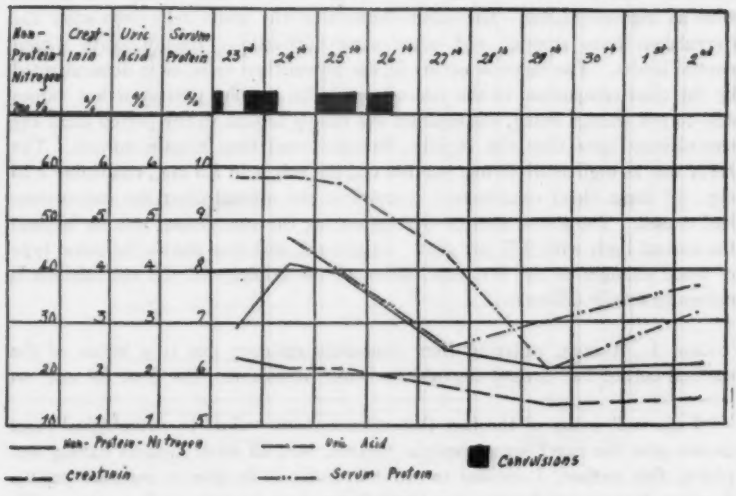

CHART 3b

Sept. 24 he had ninety-three; on Sept. 25, thirty-four; on Sept. 26, eighteen convulsions. Again, the character of the blood-changes is the same as in the first two cases. During the convulsions the leucocytes and polynuclear cells are increased; lymphocytes and eosinophiles are decreased. After the convulsions leucocytes and polynuclear cells drop to the normal, whereas lymphocytes and eosinophiles rise to the normal. Uric acid and creatinine are already increased through the first convulsions, whereas NPN, which generally rises more slowly, according to its nature as a complex compound, shows a gradual increase during and as a result of the continued attacks. After the convulsions have ceased all these constituents as well as the serum protein drop to normal values.

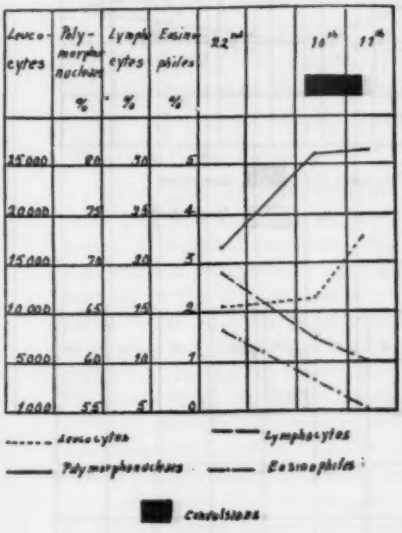

CHART 4a

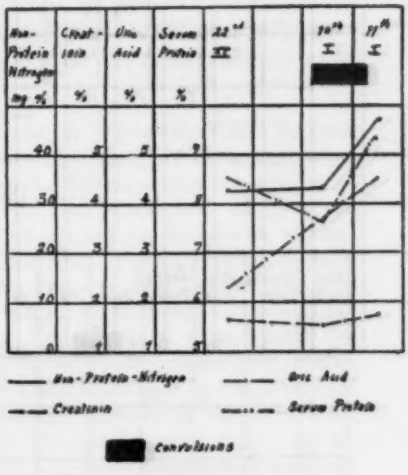

CHART 4b

The curves of this case with still another etiology of the convulsions show in general the same behavior as in the first two cases. It may be noticed that in this case, although the convulsions have been far more numerous than in the second case, the changes do not reach the same height as in that case. This is probably due to the individual constitution and capacity for reaction.

This last case showed how the NPN was raised by the continuous convulsions. The same behavior is demonstrated by Case 4.

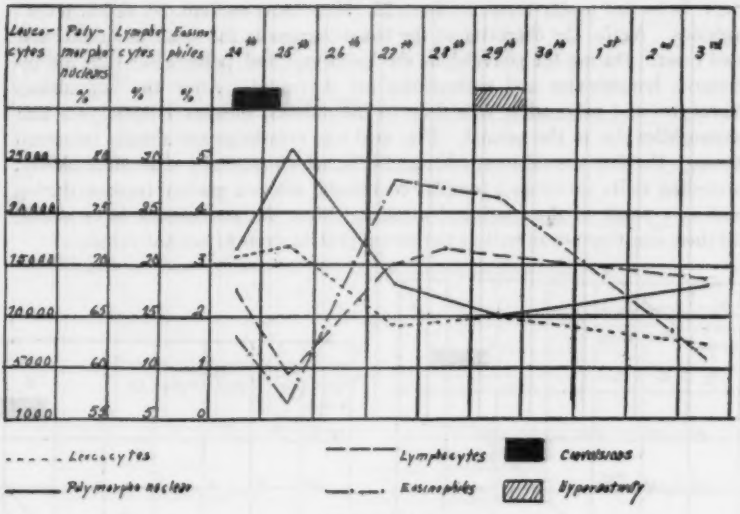

CHART 5a

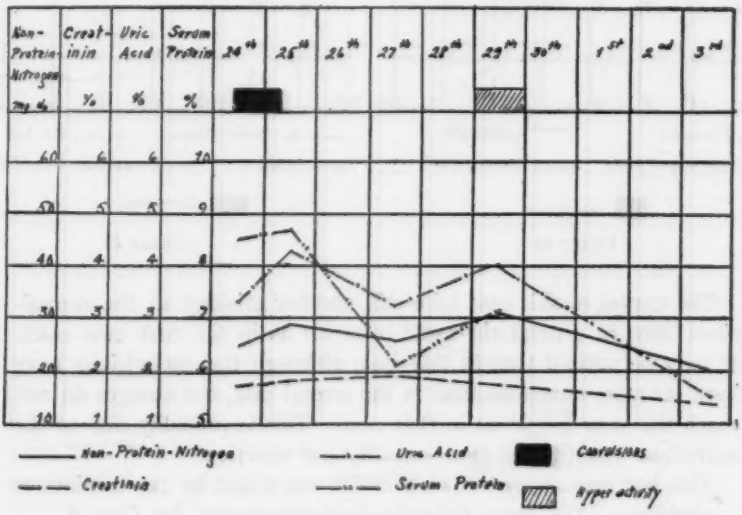

CHART 5b

Case 4. A female, suffering from general paresis with convulsions. The patient was examined on admission, Nov. 22. Two months later, on the 10th of January, she developed convulsions and had 27 attacks. Blood was taken immediately after the onset of convulsions and towards the end of the convulsive series. The curves show clearly how the total leucocyte count and the polynuclear count increase and how lymphocytes and eosinophile cells are decreased during the convulsions. NPN, uric acid, creatinine, and serum protein rise during the convulsions.

Whereas the other cases showed a return of the changes from the height during the convulsion to the normal level, this case demonstrates the reverse process, the change from the normal to the high findings during convulsions. In this case as well, although the convulsions are again of a different etiology, and a different direction of the changes was noted, the changes show the same tendency as in the first three cases.

Case 5. One of genuine epilepsy in a male (H. W.) again presents the same facts with which we have already become acquainted, but shows some other points of interest. This patient had four convulsions during the morning of the 24th and two more in the night of the 24th to the 25th. He then was clouded; on the 29th he showed a state of confused excitement with psychomotor overactivity; then he became clear and quiet. The total number of leucocytes after four attacks was 15,800, and after six convulsions 16,800; they then dropped to normal, but showed another though not so marked increase on the 29th, after which they dropped definitely. The polynuclear curve followed the curve of the leucocytes except on the 29th, on which occasion there existed a relative lymphocytosis. Lymphocytes and eosinophile cells showed a decrease during the convulsion, an increase exceeding the normal after the convulsions, and a subsequent fall to the normal. NPN, uric acid and serum protein showed an increase during the convulsions; after the convulsions stopped they decreased but rose again on the 29th, after which they returned definitely to the normal limit; the creatinine, which is not as fine an "indicator," also showed an increase during convulsions, and after their cessation a slow decrease.

The same behavior is shown in *Case 6* (R., a case of genuine epilepsy in a male), with the exception that only the uric acid showed an increase during the convulsions. This is probably due to the fact that uric acid seems to be the blood constituent which is most difficult for the kidney to excrete and which therefore forms a more sensitive indicator of any retention or incomplete elimination.

This patient showed during and after the convulsions the same

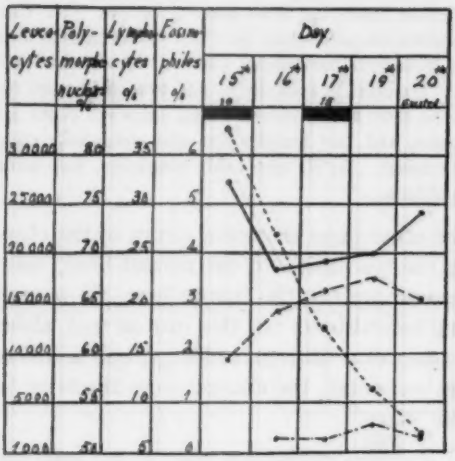

CHART 6a

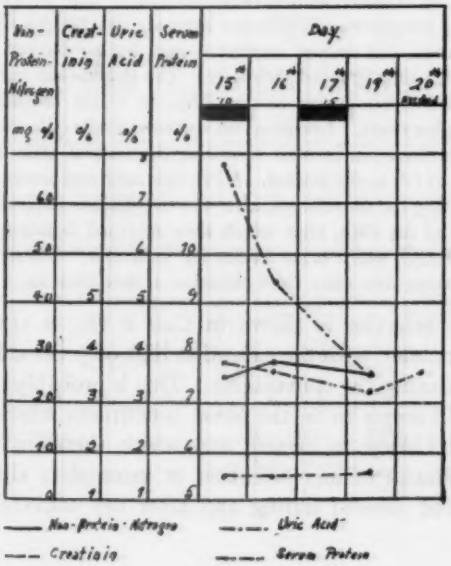

CHART 6b

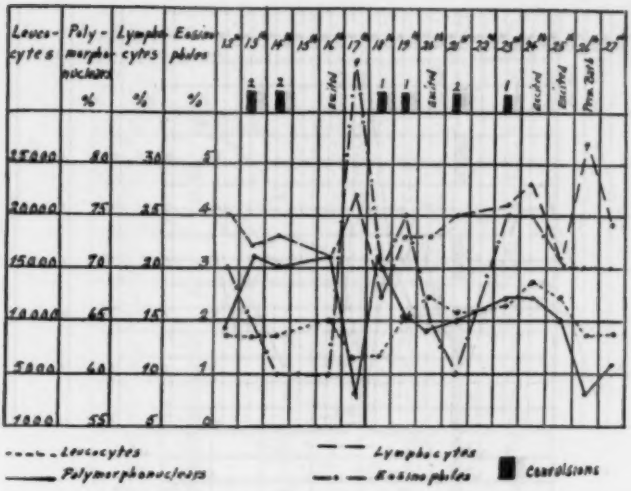

CHART 7a

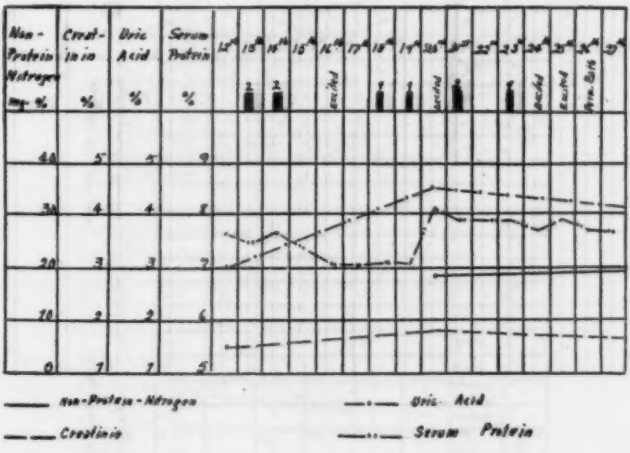

CHART 7b

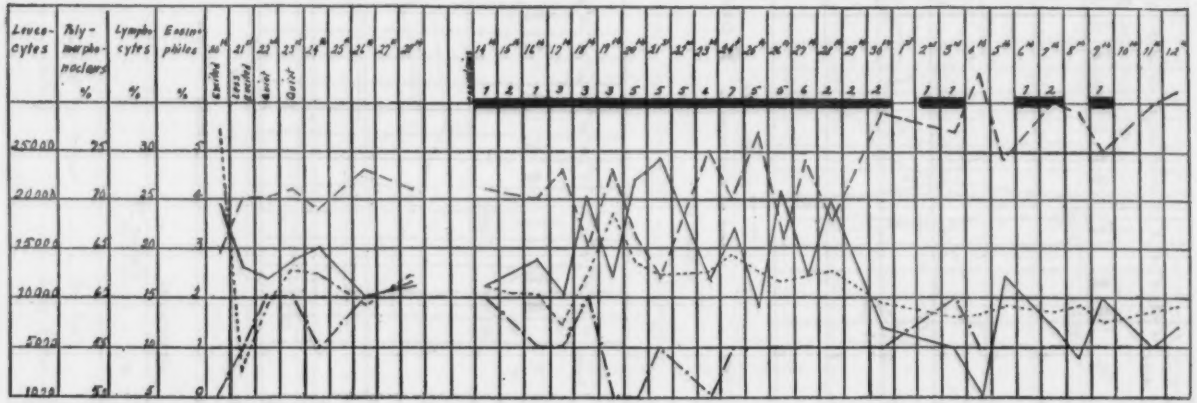
Leucocytes
Polymorphonuclears
Lymphocytes
Eosinophiles
convulsions

CHART 8a

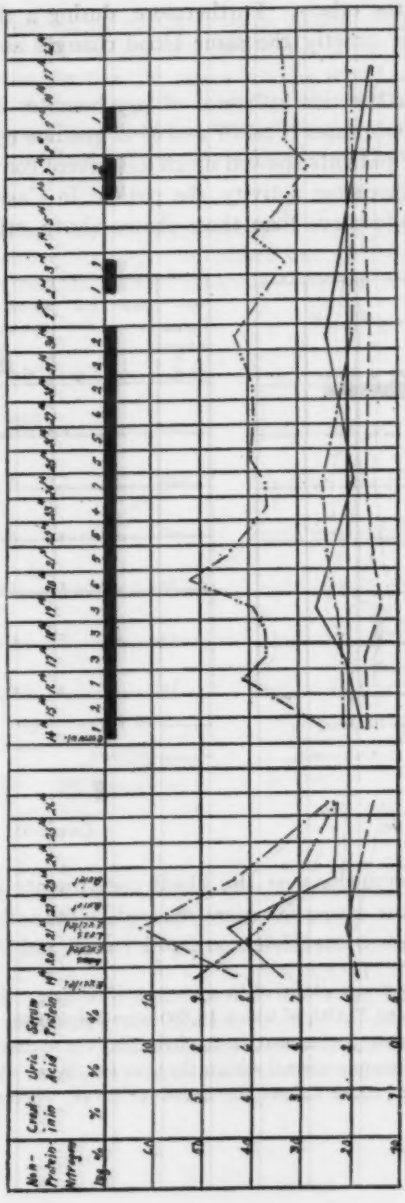
Non-Protein Nitrogen Mg. %
Creatinin %
Uric Acid %
Serum Protein %
Non-Protein-Nitrogen
Creatinin
Uric Acid
Serum Protein
convulsions

CHART 86

blood changes as the others. Furthermore, during a time of overactivity he showed exactly the same blood changes as during convulsions.

To illustrate further this influence of psychomotor overactivity, I present the next two cases (*Cases 7 and 8*) of genuine epilepsy, both in females. These patients showed much less severe convulsions and more marked psychomotor activity, the patient in Case 7, giving a far less characteristic curve than those shown above, still shows the

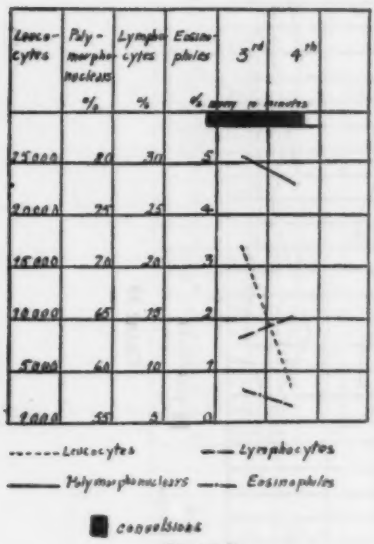

CHART 9a

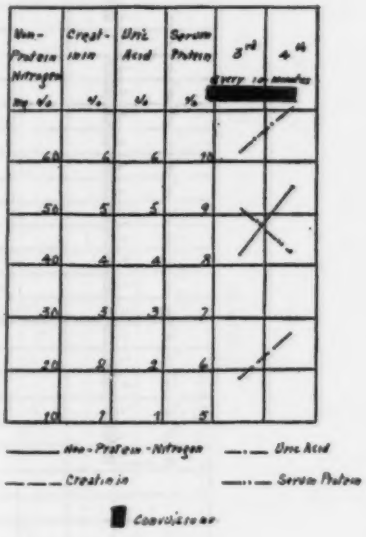

CHART 9b

influence of the convulsions on the blood constituents in the same sense as in the other cases; far more marked is the influence, however, of psychomotor overactivity on April 16, 20 and 24.

Case 8. This patient was admitted in a very excited state. The leucocytes showed an increase up to 27,100, of which 18,860 were polynuclear cells. Under the influence of treatment (permanent baths, hyoscine) the excitement subsided and the blood picture became normal within the next few days. We may notice that the serum protein curve follows the leucocyte curve, whereas NPN, uric

acid and creatinine lag behind, so to speak, increasing during excitement and falling gradually; this behavior is evidently due to the time needed for these constituents to wander from the tissues into the blood and for their excretion with the urine. The second part of the chart demonstrates only a higher level and a certain instability of the blood constituents during the period of frequent attacks with a subsequent decrease and stabilization in a period during which convulsions are less frequent.

Case 9. This case, one of genuine epilepsy in a female, shows totally different and very interesting changes. The patient had for some time at her home been suffering from convulsions, occurring every ten minutes. She was ad-

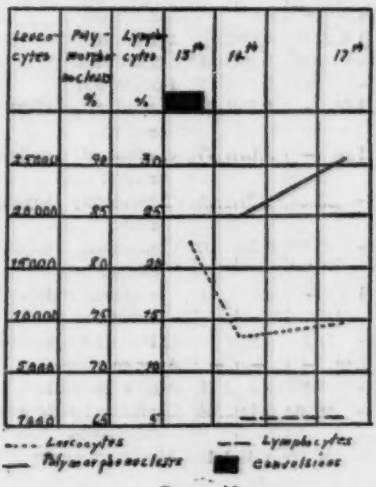

CHART 10a

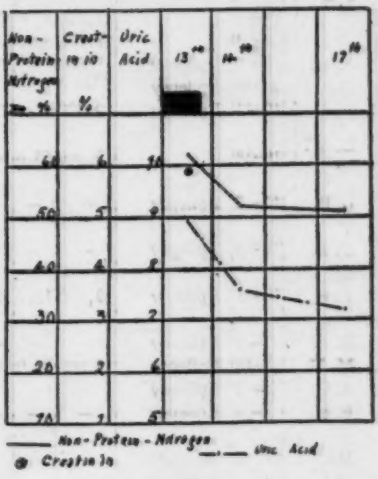

CHART 10b

mitted on Dec. 3 at noon, and died on Dec. 4, at noon, having had convulsions every 10 minutes up to the time of her death. In this case, the leucocytes having increased up to 16,900 through the previous convulsions, showed no further increase during the later ones, but a drop to 3,500 shortly before death; the polynuclear cells fell from 13,570 to 2,750, whereas the lymphocytes showed less change and the eosinophile cells remained low throughout. NPN, uric acid, and creatinine showed a steady increase, whereas the serum protein decreased. Probably the falling of the protein content and the leucocytes is due to a falling of the blood pressure which has its final cause in the exhaustion of the body energy.

Case 10. G., a man suffering from cardio-vascular-renal disease with alcoholism, developed convulsions preceding an attack of delirium tremens. He shows

TABLE II

NAME	DATE	DIAGNOSIS	LEUCOCYTES	POLYMORPHONUCLEARS	LYMPHOCYTES	EOSINOPHILES	NON-PROTEIN NITROGEN	URIC ACID	CREATININE	REMARKS
				per cent	per cent	per cent	mgm. per cent	mgm. per cent	mgm. per cent	
I. G.	1–20	Epilepsy	16,000	88.3	9.3	0.6	26.68	5.49	1.53	Night, 8 convulsions; morning, 1
	1–21		12,900	81.0	11.0	0.3	—	3.09	—	1 convulsion
	2–23		7,700	68.0	26.3	0.6	20.36	3.60	1.53	1 convulsion
F. K.	3– 6	Epilepsy	—	—	—	—	61.60	5.50	1.80	1 convulsion
M.	12–10	Epilepsy	—	—	—	—	61.99	3.54	1.83	1 convulsion
H. W.	10–11	Epilepsy	9,300	61.3	28.3	3.0	—	4.50	1.83	Confusional, slightly excited
	10–14		8,200	65.6	24.3	2.0	—	5.07	1.77	Confusional, slightly excited
L. B.	11– 2	Epilepsy	—	—	—	—	33.38	4.77	1.26	Confusional, slightly excited
C. B.	11–29	Epilepsy	—	—	—	—	36.93	4.20	2.01	Confusional, slightly excited
L. B.	10– 1	Epilepsy	9,000	58.3	30.6	3.6	—	4.86	1.86	Confusional state after convulsion
K. S.	5– 1	Epilepsy	—	—	—	—	51.8	3.9	1.2	12 hrs. after convulsion
M. H.	2–18	Epilepsy	13,000	76.0	16.0	1.0	—	—	—	After convulsion
A. O.	8– 1	Epilepsy	—	—	—	—	75.95	11.4	2.28	After 5 convulsions
F. H.	8– 7	Epilepsy	—	—	—	—	48.30	6.75	2.04	Confusional state after convulsions
M.	6–13	General paresis	—	—	—	—	24.16	6.35	2.10	After 5 convulsions
S.	5–28	General paresis	—	—	—	—	26.96	6.40	1.83	After convulsion
G. S.	4–14	General paresis	12,000	—	—	—	32.97	7.20	1.09	After convulsion
K. O.	1– 8	C. N. S. lues	12,000	88.0	8.0	0	70.75	6.6	1.8	10 min. after last of 6 convulsions
G. R.	4–15	Arteriosclerosis	—	—	—	—	47.88	5.25	1.26	Confusional state after convulsion

the same changes of the blood constituents during convulsions as in the other cases, except that the changes in this case start from and return to a higher individual level, the latter being produced by the kidney insufficiency.

The cases in which only one blood sample was taken are given in Table II.

All these patients, as well as those that were examined several times, demonstrate that during convulsions the blood constituents show marked changes. Although, naturally, single examinations are not so decisive as repeated ones, yet as a whole they give sufficient evidence as to the specific tendency of the changes.

With regard to the cellular elements of the blood, we find that during convulsions there exists a tendency to an increase of the leucocytes, mostly due to an increase of the polynuclear cells, whereas the eosinophiles are generally diminished in number, and often disappear entirely; the lymphocytes show low numbers during convulsions, although sometimes during convulsions or psychomotor over-activity a relative lymphocytosis may prevail. Individual differences exist, and also differences in different convulsions in the same individual. But the general tendency seems clear and there is no indication for regarding lymphocytosis, when it is found, as an expression of a certain endocrinologically determined constitution type, as some authors have done. The readjustment of the disturbances requires some time, which also varied in individual cases, and even in the same case in different convulsive attacks. The curves generally show an over-compensation—especially on the part of the eosinophile cells—with a subsequent decline to the normal after some oscillation. Without doubt the disturbances run parallel to a certain degree with the severity of the convulsions; we also notice individual differences which seem to be due mainly to constitutional and individual susceptibility; nevertheless, severe states such as status epilepticus generally show the most marked blood changes. As in light cases the fluctuations sometimes do not exceed the ranges which according to the present day conception must be called normal I should like to emphasize not so much the absolute numbers as the general direction in which the changes lie.

The chemical constituents of the blood are also altered during convulsions. Non-protein nitrogen, uric acid, creatinine, and serum protein show an increase of varying degree. Often, especially in light attacks, they do not exceed, or at least not much, the so-called normal limits, but they do exceed what we could call the normal

individual limit, as can be shown by comparing these values with those that are normally to be found in the same individual. The serum content of non-protein nitrogen we have found to be as high as 75.95 mg. per 100 cc.; in the same case we found a uric acid content of 11.4 mg. per 100 cc. and a creatinine content of 2.28 mg. per 100 cc. It is important to bear in mind that such high values can be produced by convulsive reactions alone, in order to avoid mistaking such cases for uremia; however, in uremia, with nitrogen retention and insufficiency of the kidney there would probably also exist hypertension in the intervals between the convulsions. It may be added in this place that other investigations not yet completed have shown that the high values for NPN, uric acid, and creatinine during convulsions are due not only to a retention,[3] but also to an increased disintegration of body material.

Furthermore, we can see from the charts that the same blood-changes take place in convulsions of totally different origin: convulsions in "idiopathic" epilepsy, in traumatic epilepsy, in lues of the brain, in arteriosclerosis, in general paresis, in cardio-vascular-renal disease, show exactly the same alterations of the blood constituents. These alterations are therefore in no way specific for epilepsy or any other "disease." Theories regarding epilepsy as a disease entity which are based on these blood-changes will have to be abandoned. The changes of the blood-cells have been regarded as a sign of auto-intoxication similar to those in anaphylactic shock (de Crinis, Di Gaspero). The increase of some of the chemical blood-constituents has been the basis for the theory that epilepsy is due to an endocrinologically precipitated disintegration of body-material or to a retention of poisonous end-products of metabolism (Frisch). Since the changes, however, are not specific for epilepsy, but occur in convulsive reactions of all types, such theories would have to be restricted to the convulsive reaction itself. The charts show also that these above mentioned blood-changes do occur not only in convulsions but as the result of any marked overactivity;[4] hence, the above-mentioned

[3] This retention is a consequence of a transitory renal insufficiency, probably of vasomotor origin, which is very common in convulsions.

[4] The results of my examinations of excited patients point in this direction, and Rakestraw also found that normal individuals show the same blood changes as a result of physical exertion, which Bauer calls physical signs of fatigue.

theories would have to be related to all physical exertion, which however, seems to render them useless. One general alteration we know to be common to all pronounced physical exertion and that is acidosis of a more or less marked degree. This may be a generic condition for the genesis of some alterations, especially of the cellular elements (Hawkins); further investigation of this point seems necessary. If we regard the blood changes as the result of physical exertion, it is easily understood why the changes of the cellular elements of the blood appear and disappear more quickly than the alteration of the chemical blood-constituents, because some time will always elapse before the waste products wander from the tissues into the blood, and before they are excreted by the urine. It must be remembered that we can only discuss the behavior of the blood constituents which we have examined; I am aware of the fact that in the additive compound of the "non-protein nitrogen" the single constituents may vary without the total value of non-protein nitrogen being changed.

SUMMARY

Cases of convulsive states in epilepsy and other "diseases" have been shown to present marked changes in the cellular and chemical blood constituents.

During convulsions we observe polynuclear leucocytosis and eosinopenia with subsequent oscillations of the curve and definite decline to the normal individual level; the lymphocytes vary without any definite rule. Non-protein nitrogen, uric acid, creatinine, and serum protein show an increase during or shortly after convulsions.

Decrease of leucocytes and of serum protein content combined with increase of the chemical blood constituents in status epilepticus seems to be a bad prognostic symptom.

The changes are not always uniform in the same or in different individuals.

The changes are not specific for any disease.

The changes are not specific for the convulsive reaction, as they may be found as the result of *all* physical exertion.

To a certain degree the changes run parallel with the intensity of physical overactivity.

The described changes can be regarded as a secondary phenomenon due to convulsions or physical exertion.

The theories regarding the etiology of epilepsy or the mechanism of the convulsive reaction which are based on the changes of the above mentioned blood constituents will have to be abandoned.

No conclusions can be drawn from the cell-count or the changes of the cellular elements as to the "epileptic" representing a certain constitution type.

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THE ARTERIOMESENTERIC OCCLUSION OF THE DUODENUM

An Anatomical Study[1]

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A previous paper by the writer (*Virchow's Arch.*, 1923, ccxliv) dealt with the fundamental anatomy of arteriomesenteric occlusion of the duodenum. This report was based on clinical histories with postmortem examinations of two such cases, and on anatomical studies of the normal relationship of the abdominal organs in more than three hundred postmortem cases.

In this former communication it was emphasized that: (1) In this type of duodenal occlusion, the obstruction of the duodenum is caused by a fold of the mesentery, belonging to the part of the small intestine which is displaced into the pelvis minor. (2) Arteriomesenteric occlusion of the duodenum is of the mechanical but not of the strangulation type of ileus. (3) This condition has an opportunity of occurring in the presence of peritoneal abnormalities such as may be caused by the failure of certain peritoneal layers to fuse during development or by sundry other conditions which facilitate the descent of loops of the small intestine into the pelvis minor. (4) The muscle of Treitz (M. suspensorius duodeni) holds the caudal end of the duodenum in position. Were it not for this muscle the duodeno-jejunal junction might drop, and turn aside in such a way that it would not be possible for a fold of the mesentery to obstruct the duodenum. (5) The point of obstruction is at the pars horizontalis inferior duodeni (proximal to the flexura duodeni sinistra) and not at the flexura duodenojejunalis, as is frequently stated in the literature. (6) Although branches of the superior mesenteric artery lie between the leaves of the mesenteric fold causing the obstruction, the latter is not produced by the trunk of this vessel, nor is its site at the point where the artery

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leaves the aorta. (7) The angle between the aorta and superior mesenteric artery is at the level of the first lumbar vertebra; whereas the part of the duodenum where the obstruction occurs is at the level of the third lumbar vertebra. (8) Within the angle between the aorta and the superior mesenteric artery (the upper aortic-mesenterial-angle) is usually situated the left renal vein and never the part of the duodenum in question. If traction is applied to the superior mesenteric artery, the left renal vein may be compressed (within the angle between the aorta and superior mesenteric artery). Such compression may cause circulatory changes in the left kidney resulting in albuminuria. If, however, the venous drainage of the left kidney is anomalous (dorsal position of the vein, etc.),[2] albuminuria will not result.

The two cases reported by the writer demonstrated that arteriomesenteric occlusion of the duodenum may occur either as a primary condition followed by paralysis and passive dilatation of the stomach (case 1), or as a secondary condition following gastroplegia and dilatation of the stomach (case 2). Fig. 1 is an unpublished photograph taken of the writer's second case. The figure shows a part of the dilated stomach and the greatly dilated pars horizontalis superior duodeni. In this case a postoperative gastroplegia and dilatation of the stomach were followed by arteriomesenteric occlusion of the duodenum.

The anatomical findings in a new case of arteriomesenteric occlusion of the duodenum will be presented in this paper. The case was recently discovered in the course of routine work with students in the dissecting room of this laboratory. The anatomical findings recorded seemed to bear out the conception of the writer in his previous communication as to the etiology and anatomy of this condition and will therefore be given in some detail.

For a clearer understanding of the underlying anatomical conditions, it was found necessary to use in the description a new and more accurate terminology for the different parts of the duodenum in addition to the usual one (B. N. A). This will be presented briefly before describ-

[2] Anomalies in the number and relationships of the left renal vein were encountered by the writer in 27 out of 150 cases recently examined (18 per cent). A detailed description of the different types will be given in a later communication.

ing the case. The B. N. A. nomenclature for the various subdivisions of the duodenum is based solely on the shape of the organ. Accordingly, the duodenum is subdivided into a (1) pars horizontalis superior, (2) pars descendens, (3) pars horizontalis inferior and (4) pars ascendens. The various flexures similarly are termed (1) the flexura duodeni superior (between the pars horizontalis superior and pars descendens) and (2) the flexura duodeni inferior (between the pars descendens and pars horizontalis inferior). The term "flexura duodeni sinistra"[3] was suggested by the writer to designate the curve between the horizontal and the ascending part of the duodenum. It is important to emphasize that this curve is found to be present in all cases, if proper technic in examination is applied. The new terminology subdivides the duodenum more accurately, takes into account the peritoneal relationships of the organ and has the further advantage of giving a great deal of information concerning the development of its topographical relationships. The duodenum is thus subdivided by the radix mesocolica into an upper portion, pars supramesocolica, and a lower portion, pars inframesocolica (Huntington, 1903). The pars inframesocolica is further divided by the radix mesenterii into the pars inframesocolica dextra and the pars inframesocolica sinistra. From these the pars inframesocolica dextra is the most secluded portion of the duodenum (pars tecta [Grosseri]). It is situated between the radix mesocolica and the radix mesenterii and is covered by the mesocolon ascendens, in addition to its peritoneal sheath, as are the other parts of the duodenum below the mesocolon transversum. Hence the name pars retromesocolica.

PRESENTATION OF CASE

The clinical history of the man (58 years old, white) who died with cancer of the palate, does not record any symptoms or signs which would have suggested the postmortem anatomical findings, by which the diagnosis of an arteriomesenteric occlusion of the duodenum was made.

In Fig. 2 the organs are shown *in situ* after the abdomen was opened.

[3] The full name of this curve would be: flexura duodeni inferior sinistra. However, since the duodenum has but one flexura on the left side, the word "inferior" should be left out.

The stomach was somewhat enlarged and shifted towards the pelvis. The curvature minor with a part of the omentum minus could be seen without the usual procedure of first lifting the liver. The lowest point of the curvatura major was below the umbilicus. The transition of the pars pylorica into the pars horizontalis superior duodeni was gradual and the latter was not collapsed. The liver was not enlarged and did not project below the costal margin. The fundus of the gall-bladder was adherent to the great omentum and to the duodenum.[4] At the entrance of the small pelvis lay the transverse colon (only

[4] Ancel and Sencert (1903) described the ligamentum cysticoduodenoepiploicum which they found in 29 per cent of 124 adults examined. Broman (1904) agrees with these authors that the presence of this ligament does not indicate a pathological condition of gall-bladder or duodenum, because he identified it in a 49 cm. fetus and also in adults in whom the layers of the great omentum have failed to fuse. Our case confirms the observation of Broman.

FIG. 1. A CASE OF POSTOPERATIVE GASTROPLEGIA WITH DILATATION OF THE STOMACH FOLLOWED BY ARTERIOMESENTERIC OCCLUSION OF THE DUODENUM

The figure shows a part of the dilated stomach and the greatly dilated pars horizontalis superior duodeni.

FIG. 2. PRESENT CASE

Organs shown *in situ* after the abdomen was opened

FIG. 3. DIAGRAM SHOWING THE EMPTY AND COLLAPSED PARS INFRAMESOCOLICA SINISTRA

The colon transversum is reflected and its mesocolon stretched, and the loops of the jejunum with its mesentery, which did not participate in causing the obstruction, are displaced to the right.

FIG. 4

The pars inframesocolica dextra, or pars tecta [Grosseri], is made visible by lifting up the colon transversum and stretching the mesocolon. The loops of the small intestine have been displaced to the left.

FIG. 5. DIAGRAM SHOWING THE ENTIRE PARS INFRAMESOCOLICA OF THE DUODENUM MADE VISIBLE BY REMOVAL OF INTESTINE AFTER CUTTING THE MESENTERY NEAR AND PARALLEL TO THE RADIX MESENTERII

FIG. 6. DIAGRAM SHOWING THE "PRIMARY" RETROPERITONEAL ORGANS

The upper aortic-mesenterial angle contains the left renal vein. This was double and both veins united just before crossing the aorta to empty into the vena cava inferior.

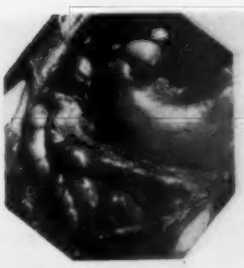

Fig. 1

Fig. 2

Fig. 3

Fig. 4

Fig. 5

Fig. 6

partly visible) covered by great omentum. By lifting the great omentum it was readily seen that its ventral and dorsal layers were imperfectly fused. Since the fusion took place only near the left and right flexurae coli and on the lowest portion of the omentum, no pars gastrocolica[5] of the omentum majus was present.

Fig. 3 shows the pars inframesocolica sinistra duodeni and also the point where the occlusion took place. To obtain this picture the colon tranversum was reflected and its mesocolon stretched. The loops of the jejunum, the mesentery of which did not participate in causing the obstruction, were also displaced to the right.

The duodenum was collapsed and empty to the left of the point where the fold of the mesentery causing the obstruction crossed the pars horizontalis inferior. This empty portion of the duodenum did not correspond exactly to the pars inframesocolica sinistra, since the point of obstruction did not occur along the line of the radix mesenterii, where this crossed the pars horizontalis inferior duodeni. The arteria mesenterica inferior marked the level of the third lumbar vertebra, where the pars horizontalis inferior duodeni crossed the vertebral column.

The radix mesenterii began as usual at the level of the lower surface of the 1st lumbar vertebra and ran parallel with, and to the right of, the pars ascendens duodeni. The collapsed part of the duodenum began at the point where the fold of the mesentery crossed the pars horizontalis inferior duodeni. This was to the right of the flexura duodeni sinistra, as the figure clearly shows. Both the pars ascendens and the flexura duodenojejunalis were to the left of the radix mesenterii and beyond the point of the obstruction. This topographical arrangement indicated anew that it is anatomically impossible for an arteriomesenteric duodenal occlusion to take place at the flexura duodenojejunalis.

In Fig. 4 the pars inframesocolica dextra (or pars tecta [Grosseri]) was made visible by lifting up the colon transversum and stretching the mesocolon. The loops of the small intestine were also displaced to the left. The visible part or the duodenum was distended up to the

[5] The pars gastrocolica (or ligamentum gastrocolicum) is the part of the ventral layer of the great omentum between the curvatura major of the stomach and the taenia omentalis of the transverse colon.

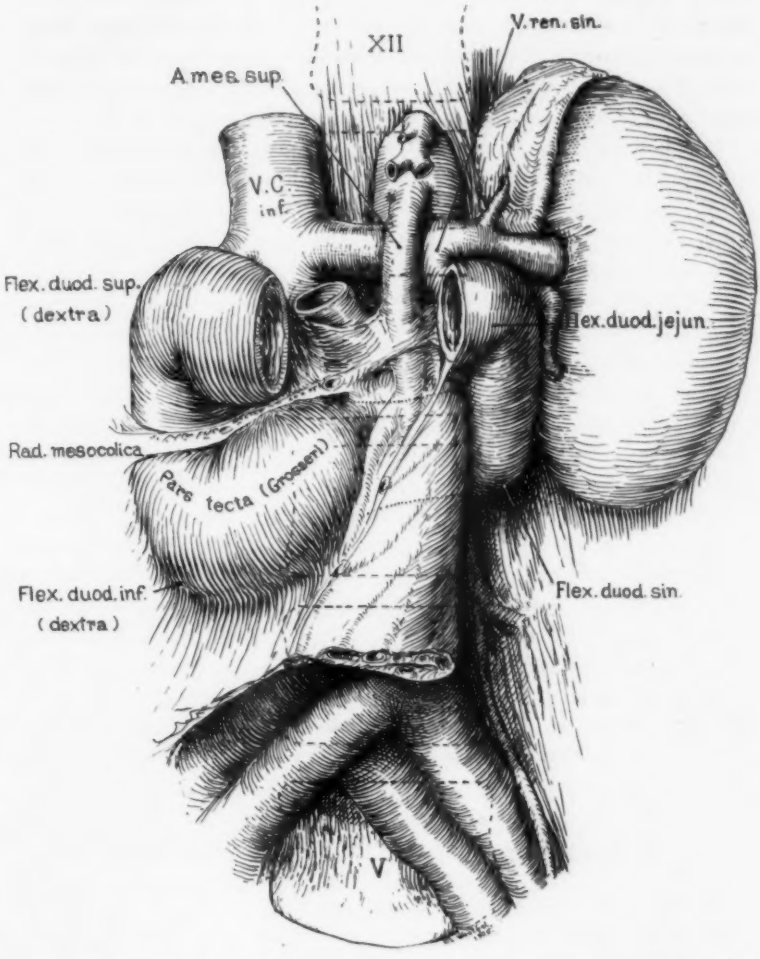

FIG. 7. DRAWING FROM DIFFERENT STAGES IN THE DISSECTION OF THE ACTUAL SPECIMEN, SHOWING THE TOPOGRAPHICAL RELATIONSHIPS OF THE DUODENUM, THE LEFT RENAL VEIN AND THE SUPERIOR MESENTERIC ARTERY

The flexura duodenojejunalis is shifted downward in order to show the vena renalis sinistra in the angle between the aorta and the superior mesenteric artery.

point where the radix mesenterii (or better the fold of the mesentery causing the obstruction) crossed the pars horizontalis inferior duodeni.

In Fig. 5 the entire pars inframesocolica of the duodenum was made visible by cutting the mesentery near and parallel to the radix mesenterii and removing the small intestine. Previous to this the muscle of Treitz was carefully dissected. A fan-shaped group of fibers was found to be inserted by its broad end into the terminal portion of the pars ascendens duodeni and to converge to an apparent origin about the trunk of the superior mesenteric artery and the coeliac axis. At the origin these fibers radiated out in different directions. The size, form and insertion of the muscle were identical with those of other similarly prepared cases.[6]

The arteria and vena mesenterica superior were prepared with some of their branches by removing the peritoneum on the right side of the remaining mesentery. A number of branches left the superior mesenteric artery, appreciably reducing the size of the trunk before it reached the point in the duodenum where the obstruction occurred. These branches were found to be: the A. pancreaticoduodenalis inferior, the A. colica media on the right, and four (or five) of the Aa. jejunales on the left.

The ileocaecal part of the gut (caudal end of the ileum, the caecum and appendix) was noted in the right fossa iliaca and was not displaced into the small pelvis.

In Fig. 6 the organs are seen situated behind the peritoneum ("primary" retroperitoneal organs).[7] The upper aortic-mesenterial-angle contained the left renal vein. This was double and both veins united just before crossing the aorta to empty into the vena cava inferior.

[6] A special study of the folds about the flexura duodenojejunalis, considering especially the nature of the muscle of Treitz and the relation of the folds to this muscle and to the vena mesenterica inferior, is under way at present.

[7] Organs situated originally behind the peritoneum in the cavum retroperitonei and which have never had a peritoneal covering are "primary" retroperitoneal organs (e.g., kidneys). The organs which lose their peritoneal covering on one side during development are "secondary" retroperitoneal organs (*e.g.*, colon ascendens). If such a secondary retroperitoneal organ becomes covered again with peritoneum during development, while another organ loses its peritoneal suspension, then we speak of a "tertiary" retroperitoneal organ (*e.g.*, pars retromesocolica duodeni).

Fig. 7 is a drawing combined from different stages in the dissection of the actual specimen. It shows schematically the topographical relationships of the duodenum, the left renal vein, and the superior mesenteric artery. The radix mesocolica is indicated and is seen to separate the duodenum into an upper and a lower part. The pars supramesocolica duodeni consists of the pars horizontalis superior and a part of the pars descendens, curving over into each other at the flexura duodeni superior. The pars supramesocolica duodeni is shown in the figure to be only moderately dilated and to be shifted a little downwards. Below the radix mesocolica the fold of the mesentery separates the dilated portion of the duodenum from the rest of it by crossing the pars horizontalis inferior. On the left side of the fold of the mesentery causing the obstruction, the empty parts of the duodenum are seen. These do not exactly correspond to the borderline between the pars inframesocolica dextra and sinistra. To the pars inframesocolica sinistra belong: (1) the pars horizontalis inferior duodeni, from the radix mesenterii up to the flexura duodeni sinistra; and (2) the pars ascendens duodeni, which curves over at the flexura duodenojejunalis into the jejunum.

The flexura duodenojejunalis is shifted downwards in Fig. 7 in order to show the vena renalis sinistra in the angle between the aorta and the superior mesenteric artery. This figure also demonstrates that the radix mesenterii runs parallel to, and to the right of, the pars ascendens duodeni. Because of this anatomical arrangement and the topographical relationship between the superior mesenteric artery and the flexura duodenojejunalis, an arteriomesenteric occlusion of the duodenum is impossible at this point, that is, at the flexura duodenojejunalis. The obstruction in such a case is always, as said before, in the pars horizontalis inferior duodeni and proximal to the flexura duodeni sinistra.

DISCUSSION

It is difficult to say whether the case here presented is an example of the acute or the chronic type of arteriomesenteric occlusion of the duodenum because of insufficient clinical history, preservation of the body and the time interval between death and anatomical examination. The distinction between the acute and the chronic types of this

condition is, however, from an anatomical point of view, of minor importance, since they both have the same causal and formal genesis. In both cases a fold of the mesentery belonging to a part of the small intestine, which is displaced into the pelvis minor, prevents the normal patency of the duodenum at a point proximal and near to the flexura duodeni sinistra. At this point the pars horizontalis inferior is flattened in a ventro-dorsal direction, the compression occurring between the above mentioned fold of the mesentery on the one hand, and the aorta or vertebral column on the other. The bulging of the lumen of the duodenum at this point, when necessary for the normal passage of the contents of the bowel, is thus either made difficult or prevented entirely. In the chronic type where the occlusion is incomplete, patency is apparently obtained intermittently by increased peristaltic force in the more oral portion of the gastro-intestinal tract. If the increased contraction exerts insufficient force to propel the contents past the critical point, the occlusion becomes constant and complete. It is evident, then, that the intermittent and incomplete (or chronic) type of arteriomesenteric occlusion of the duodenum may at any time become constant and complete (acute).

A constant opportunity is offered for the occurrence of this condition, especially in the presence of peritoneal abnormalities, which facilitate descent of the small intestine into the pelvis minor, providing a possibility for the mesentery of the displaced loops to form a fold across the duodenum. In our case the fusion of the ventral and dorsal layers of the great omentum did not take place along the taenia omentalis of the transverse colon as is usual; a deep recessus inferior bursae omentalis resulted and the colon transversum was found lower down in the abdominal cavity. Since this condition prevailed, the colon and the mesocolon transversum caused the loops of the small intestine to be shifted down into the pelvis providing an opportunity for the mesentery to form an obstructing fold across the pars horizontalis inferior duodeni if other circumstances were favorable (empty bowels, etc.).

For those cases of arteriomesenteric occlusion of the duodenum for which surgical intervention has been definitely decided upon, duodenojejunostomy is the procedure of choice.

This operation was first suggested by Barker at a meeting of the

Johns Hopkins Medical Society (November, 1905) in discussing Dr. Finney's paper ("The Relation of Dilatation of the Duodenum to Gastric Disturbances"). Duodeno-jejunostomy was also suggested by Bloodgood in 1906, but Stavely performed it first in December, 1907. It consists of connecting the pars inframesocolica dextra (which is just proximal to the obstruction) with the first loop of the jejunum by anastomosis. The technique of this operation is described in detail by Kellogg and Kellogg, in their paper of 1921, with a report of 41 of their own cases and 17 from the literature up to that date. The cases of Devine (3), Wilkie (21), Hurst (1), Barling (4), Tinkham (2), Griffith (6), Remijnse (4), Haberer (1), McKenty (13) and those reported recently by Weiss (34) make a total number of 147 cases found in available literature in which the Barker-Stavely operation has been performed.

Bloodgood was the first (1907) to demonstrate the association of chronic duodenal occlusion with a mobile ascending colon. Several cases of this association have been reported since (Wilkie, McConnell and Hardman, McKenty). Bloodgood (1912) suggested and carried out successfully in five such cases the resection of the right half of the colon in this condition. Others (Wilkie, McConnell and Hardman, McKenty) performed colopexy. This type of duodenal occlusion, associated with a mobile ascending colon, is not an arteriomesenteric occlusion—a conclusion reached by consideration of the underlying anatomical factors.

According to Bloodgood (1912), "The constriction of the mesentery is due to a pull on the mesentery by the dilated caecum displaced into the pelvis. This caecum cannot produce such tension on the mesentery of the small intestine unless the last portion of the ileum near the caecum has an unusually short mesentery." In the writer's experience, the last portion of the ileum always has a very short mesentery, or better, it never has one of any considerable length unless the caecum is movable. A displaced caecum can hardly exert sufficient tension on the mesentery of the *small* intestine to produce an obstruction of the duodenum, but it may do so on the mesocolon, provided that the right half of the colon has a mesocolon and is freely movable. The occlusion of this type is caused by the mesocolon and not by the mesenterium and is therefore rather an "*arteriomesocolic*" than an "*arteriomes-*

enteric" occlusion. A pulling on the superior mesenteric artery is present also in this case and is exerted by the A. ileocolica, A. colica dextra and A. colica media and not by the Aa. jejunales as in the case of arteriomesenteric occlusion.

A proper conception of the anatomical conditions underlying the "arteriomesenteric" and the "arteriomesocolic" occlusion of the duodenum is not a pure academic discussion. For those cases in which surgical intervention is indicated, the choice of the procedure depends upon the proper recognition of the type of occlusion. This has been recognized by Bloodgood who did not perform duodeno-jejunostomy, and by Wilkie who had performed this operation in twenty-one of his cases where it was indicated. McConnell and Hardman "have not performed duodeno-jejunostomy," since all of their "cases of arterial duodenal ileus had a mobile ascending colon." They performed colopexy in fourteen out of their nineteen cases, McKenty in thirteen out of his twenty-six cases. From all these it is evident that to perform the Bloodgood operation or Wilkie's colopexy (that is, fixation of the caecum and colon) in case of an arteriomesenteric duodenal occlusion is as useless a procedure as to carry out the Barker-Stavely operation (duodeno-jejunostomy) in case of an arteriomesocolic obstruction of the duodenum.

SUMMARY

The anatomical findings in a case of arteriomesenteric occlusion of the duodenum are presented.

The occlusion was caused by a fold of the mesentery, belonging to a part of the small intestine, displaced into the pelvis minor. Up to the point where this fold crossed the duodenum, the gut was found to be distended; beyond it, collapsed and emptied. The obstruction was located on the pars horizontalis inferior near the point where it curves over into the pars ascendens duodeni.

Absence of the pars gastrocolica of the great omentum was noted. Since the fusion of the ventral and the dorsal layers of the latter did not take place along the taenia omentalis, the transverse colon was found lower down in the abdominal cavity. Consequently, its mesocolon caused the loops of the small intestine to be shifted down into the pelvis, providing an opportunity for the mesentery of these loops to form an obstructing fold across the duodenum.

The difference between "arteriomesenteric" and "arteriomesocolic" occlusion of the duodenum is pointed out in the discussion and the special importance of such a distinction where surgical intervention is indicated is emphasized. For in cases of arteriomesenteric occlusion a duodeno-jejunostomy (Barker-Stavely operation) is to be considered the rational procedure, whereas resection of the caecum or of the right half of the colon (Bloodgood), or colopexy (Wilkie) is the operation of choice in treatment of arteriomesocolic duodenal occlusion.

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THE BLOOD CHEMISTRY IN ECLAMPSIA[1]

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In a study of the blood chemistry in toxemic patients in this Clinic, we have been able to detect certain very definite changes in eclampsia. It is the purpose of this paper to present briefly these variations from the normal.

As shown in a previous publication, the cation ratios in the blood of eclamptic women are within normal limits, with the possible exception of the $\frac{(P)}{(Ca)}$ ratio, which is slightly elevated. We suggested that this ratio might be associated with the hyperglycemia sometimes noted in this disease, and we also directed attention to the increased uric acid content and the low CO_2 combining power of the blood.

In the accompanying tables and graphs are given the non-protein nitrogen, sugar, CO_2 combining power, uric acid, lactic acid and inorganic phosphorus. The non-protein nitrogen was determined by the Folin-Wu method, the uric acid by Folin's and the sugar by Benedict's method. For lactic acid determination the method of Clausen was used, and that of Briggs for inorganic phosphorus.

In Tables I and II, we find the values for normal, non-pregnant and pregnant women, respectively. One notes that pregnancy is accompanied by a slight decrease in the N.P.N. and the CO_2 combining power, as has already been shown by several authors. In lactic acid, we notice no difference between the non-pregnant and pregnant patients. One is struck by the remarkable uniformity of the values, and it may be well to bear in mind that our blood samples were taken from patients resting in bed and in the morning before breakfast.

From a study of Table III, we see that eclampsia is associated

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TABLE I

Normal non-pregnant

CASE NUMBER	N.P.N.	SUGAR	CO_2	URIC ACID	LACTIC ACID
1	33.0	90	58.2	2.6	
2	33.1	87	55.7	2.1	
3	30.8	81	57.6	3.0	
4	28.5	101	57.7	2.5	27.09
5	30.0	100	54.9	3.3	36.22
6	31.0	103	54.5	3.5	24.15

TABLE II

Normal pregnancy

CASE NUMBER	N.P.N.	SUGAR	CO_2	URIC ACID	LACTIC ACID
7	27.1	100	43.2	3.2	
8	28.1	91	45.7	2.8	
9	29.2	93	46.3	2.7	
10	25.9	87	41.0	2.9	12.34
11	28.5	72	46.2	3.3	28.30
12	27.3	69	48.1	3.5	32.60
13	29.2	89	47.7	3.5	14.49

TABLE III

Eclampsia

CASE NUMBER	N.P.N.	SUGAR	CO_2	URIC ACID	LACTIC ACID	REMARKS
14	36.6	125	35.3	6.9		Stroganoff treatment with insulin
	36.4	73	46.7	3.5		Two days later
15	26.7	146	34.3	9.0		Stroganoff treatment with insulin
		108	36.3			Four hours later
16		104	19.2	8.4		Stroganoff treatment with insulin
		91	51.0			One day later
17	30.6	118	20.0	7.5	190.00	Stroganoff treatment with insulin
	29.2	85.5	51.0	3.5	39.15	One day later
18	36.4	105	36.5	6.7	120.75	Stroganoff treatment
	37.5	82	45.6	5.4	41.06	Two days later
19	54.6	145	34.0	8.8	93.60	Stroganoff treatment with insulin
	61.5	116	40.9	4.5	47.50	Two hours later

with some very definite changes in certain of the blood constituents, which may be summed up as follows:

(1) A pronounced tendency towards hyperglycemia.

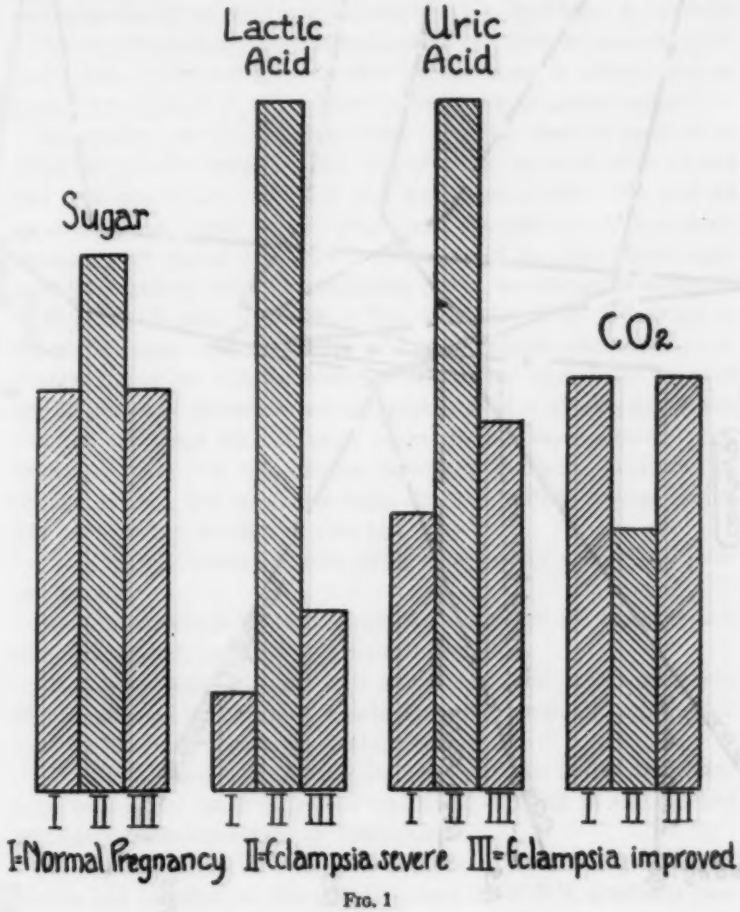

FIG. 1

(2) An increased uric acid content.

(3) An increased lactic acid content.

(4) A very low CO_2 combining power in certain cases.

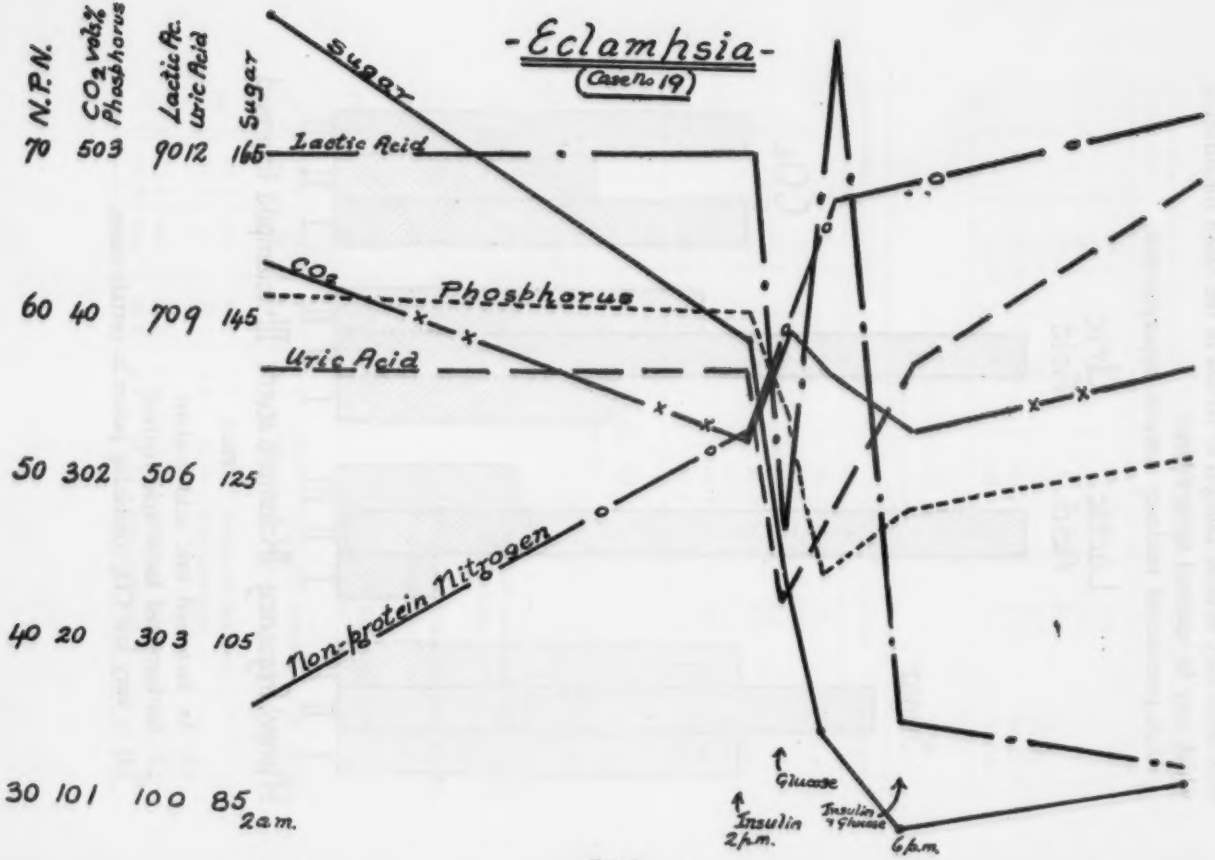
-Eclamhsia-
Case No. 19
N.P.N.
CO2 vols.%
Phosphorus
Lactic Ac.
Uric Acid
Sugar
70 503 9012 165
60 40 709 145
50 302 506 125
40 20 303 105
30 101 100 85
2a.m.
Sugar
Lactic Acid
CO2
Phosphorus
Uric Acid
Non-protein Nitrogen
Glucose
Insulin
2 p.m.
Insulin + Glucose
6 p.m.

FIG. 2

In the eclamptic table (III), we have also given the analysis on a second blood specimen from each patient, usually taken after improvement or within a few hours after the use of insulin. The remarkable change in the blood picture is shown by Figure 1, in which we have plotted the average values for the first five patients shown in Table III. It is readily seen that improvement is always accompanied by a return of these blood constituents to normal values.

One patient in the eclamptic table (Case 19) died 23 hours after admission to the hospital. She was admitted in coma after having had four convulsions at home and no prenatal care. We had an opportunity to study her in detail, and the changes in her blood chemistry are shown in Figure 2. Because of the high blood sugar and the relatively low CO_2 combining power, we deemed it advisable to give her 25 units of insulin. This was followed by a decrease in the blood sugar, uric acid, lactic acid and inorganic phosphorus, and a slight rise in her CO_2 combining power. Two hours later, we gave her 50 grams of glucose as a protection against a hypoglycemia and followed this later with 10 units of insulin and more glucose. Immediately after the first glucose injection, the lactic acid rose to about 105 mg., but gradually came down to within normal limits. The outstanding features of this case are:

(1) A marked hyperglycemia which disappeared under the use of insulin.

(2) A pronounced increase in the lactic acid in the blood, which also disappeared, probably as a result of insulin.

(3) A very high uric acid content in the blood, which was only slightly affected by the use of insulin and which gradually rose to 12.2 mg. per 100 cc. of blood, just before death.

(4) A CO_2 combining power that was slightly below the lower limit for normal pregnancy, but which did not fall to the marked acidosis level so often seen in eclamptics.

(5) A nitrogenous retention, as evidenced by the high N.P.N. Insulin had no effect on this retention, and the N.P.N. gradually rose to 75 mg. per 100 cc. of blood.

Post-mortem examination revealed a greatly damaged liver, typical of eclampsia. It is our opinion that, when the patient entered the hospital, too great liver damage had already been sustained, and

although insulin probably prevented the development of a marked acidosis with great increases in sugar and lactic acid, we were unable to overcome the rapidly developing nitrogenous retention and the gradual increase of uric acid to an exceptionally high figure.

It occurred to us that the very marked increase in lactic acid in the blood of the eclamptic patient might be due solely to the muscular activity incident to the convulsions. With this in view, we studied the conditions in a pregnant woman suffering from epilepsy, and close to term. We were successful in obtaining a blood sample from her within a few minutes after a rather violent epileptic convulsion. It showed the following:

N.P.N.	30	mg. per 100 cc. blood
Sugar	106	mg. per 100 cc. blood
Lactic acid	45.32	mg. per 100 cc. blood
CO_2 combining power	50.7	volumes per cent

These values are all within normal limits, except that the lactic acid is slightly elevated. The increases in lactic acid noted in eclamptic patients are far greater than that seen in the epileptic patient.

DISCUSSION

Zweifel, in 1904, showed that in the urine of eclamptic women the urea-nitrogen is lowered and the ammonia nitrogen raised. From this, he reasoned that there ought to be an increase in some acid in the blood, and that it was probably in lactic acid. He accordingly analysed the urine and blood for that substance, precipitating it as zinc lactate. He reported eight eclamptic cases in which he was able to show the presence of lactic acid in the blood. From one of the patients, he obtained two specimens of blood, the first showing lactic acid, while the second did not, this second sample of blood having been taken about five hours after the last convulsion. He furthermore stated that in normal pregnancy he was usually unable to demonstrate the presence of lactic acid. It might be added that Füth and Lockemann demonstrated the presence of lactic acid in the cerebrospinal fluid of eclamptic patients. Our figures, on the other hand, show that, in the blood of normal pregnant women, the lactic acid is within the limits for the normal non-pregnant person, and that in eclampsia we have a pronounced increase, amounting to 200 per cent or more above the normal.

It should be pointed out that in Case 18 in Table III, as well as in an epileptic woman, we obtained the blood specimens immediately after a convulsion, and in the former after the first fit. From this, it must be evident that, almost immediately after an eclamptic convulsion, a marked increase in blood lactic acid makes its appearance. Some may also argue that the elimination of lactic acid is greatly interfered with in eclampsia. In answer to this, we refer to our Cases 17 and 18, in which there was no retention, and the elimination was good; while the same applies to the epileptic patient.

J. L. Williams in 1921, directed attention to the increased amount of uric acid in the blood in the toxemias of pregnancy. The very marked accumulation of uric acid in eclampsia is one of our most constant findings.

That a disturbance in the liver may be the cause of the increase in lactic and uric acids is possible. In the normal individual, the major portion of lactic acid is changed back into glycogen, and this is stored in the liver and the muscles. It seems to us that there are three possibilities which may explain a high lactic acid in the blood: (1) An abnormally great amount of carbohydrate metabolized; (2) a disturbance or slowing down of the "lactic acid to glycogen" step in the carbohydrate chain; (3) lactic acid production from sources other than carbohydrate, such as protein.

The evidence at hand is not sufficient to permit us to discuss further the rôle which the liver might play in the production of the blood chemistry picture which we have described in eclampsia. It should, however, be pointed out that the work of Mann on removal of the liver suggests that the increased uric acid may be due to liver injury.

CONCLUSIONS

A. The outstanding chemical findings in the blood of eclamptic patients are:

(1) A high uric acid.

(2) A markedly increased lactic acid, not wholly due to muscular hyperactivity.

(3) A decrease in the CO_2 combining power, which is very pronounced in certain cases.

(4) A definite tendency towards a hyperglycemia, which is often associated with a high inorganic phosphorus.

B. There is usually no increase in the non-protein nitrogen of the eclamptic blood. When such an increase is present, it may be associated either with a nephritis, upon which the eclampsia has been superimposed, or with the last stages of the disease. Furthermore, there is a slight, but definite decrease in the blood urea-nitrogen, as has already been pointed out by one of us.

C. At present, we are not in a position to state in how far the eclamptic blood picture is dependent upon the lesions usually observed in the liver.

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BOOK REVIEWS

Modern Surgery. By JOHN CHALMERS DACOSTA, M.D., LL.D., F.A.C.S. 9th ed. revised with 1200 illustrations. 1527 pp. $10.00. (*W. B. Saunders Co., Phila., 1925.*)

The last edition of DaCosta's surgery differs from the preceding one chiefly in the matter of size. The style and character of the book are unchanged. As a text-book of general surgery this volume leaves much to be desired. As a handy reference book it is unsurpassed. Each succeeding edition has grown by the accretion of new data without sufficient omission of the obsolete. Fundamental surgical principles are obscured. There is not sufficient discrimination between valuable and valueless data. Frequently there is too much stress on therapeutic methods and too little on differential diagnosis. This volume does, however, contain an amazing number of facts. It has been said, and is suspiciously true, that one can find anything in DaCosta.

W. F.

Lacto-Bacillus Acidophilus. By NICHOLAS KOPELOFF. 211 pp. $5.00. (*The Williams & Wilkins Co., Baltimore, 1926.*)

Kopeloff's book is devoted to a thorough analysis of the experimental data on which sour-milk therapy is based and to "an attempt to reduce clinical observations to quantitative terms." It covers the literature on the subject; it discusses in some detail the work of Metschnikoff and Cohendy on *Lacto-bacillus bulgaricus*, which, although not the first work in this field, stimulated interest in it immensely, since Metschnikoff firmly believed that harmful intestinal bacteria were the cause of many human ills, and that better health and greater longevity would follow their replacement by his sour-milk bacillus. Kopeloff shows very distinctly, both from the studies of others and his own, that such replacement is ephemeral and that the clinical results of *Lacto-bacillus bulgaricus* have been, in the main, highly disappointing, but that in *Lacto-bacillus acidophilus* we have an organism which can be transplanted into the human intestine, which can convert the flora from a Gram-negative proteolytic to a Gram-positive fermentative type, and which can be used in a variety of chronic conditions, especially chronic constipation, with beneficial results. The basis of such conclusions is the extensive experimental work of a number of investigators, notably Rettger and his associates, and it is from the Yale University Laboratory that Kopeloff obtained the cultures for his own extensive experimental and clinical investigations. He found that relief was obtained in 54 cases of constipation by the administration of acidophilus milk—1000 cc. daily, with or without lactose, although lactose materially prolonged the beneficial effects of the acidophilus milk feeding after the cessation of its administration—while in some cases improvement seemed to be permanent

even without the use of lactose. He also had marked success with the same therapy in 13 cases of acute diarrhea—a result which has not been the experience of the reviewer, although in the constipated cases we have obtained in a number of instances satisfactory results similar to those of Kopeloff.

Kopeloff's very extensive studies of the influence of the *L. acidophilus* on the intestinal flora lead him to believe that the injection of the organism in large numbers "tends to transform the intestinal flora from a Gram-negative to a Gram-positive type," that a high relative percentage of this organism is almost invariably associated with an increase in the number of the stools, usually of softer consistence and lighter color, and that the organisms may be recovered from the stool for a long time after treatment has ceased. From the results of many experiments he feels that *L. acidophilus* therapy is essentially bacteriologic, rather than physical or chemical. A long chapter is devoted to the subject of the effect of constipation on indican production and elimination, and he finds—and in this we decidedly agree—that there is no correlation between constipation and indican urinary excretion; that it is not affected by high or low carbohydrate diets, that there is no correlation between the intestinal acid-uric flora and urinary indican; and—of interest because of the views of certain psychiatrists—that it is impossible to correlate the mental state of psychiatric patients and the indican excretion.

The volume concludes with some practical recommendations on the preparation and administration of *L. acidophilus* milk; with warnings on the worthlessness of most of the commercial tablets and many of the commercial milk preparations, but with insistence on the indubitable value of lactose in conjunction with the sour milk therapy.

The book represents a careful study, in the main, a fair review of the literature, and a very large amount of personal observations both experimental and clinical; it shows that *L. acidophilus* therapy has a scientific basis and that it has been useful in certain intestinal conditions—and we can recommend it heartily as really the most thorough exposition of the entire subject which has appeared up to the present time.

T. R. B.

Food for the Diabetic. By MARY PASCOE HUDDLESON. With an Introduction by Nellis Barnes Foster, M.D. 2nd ed. 78 pp. $1.25. (*The Macmillan Co., N. Y.*)

The author has aimed to give a practical understanding of Diabetes and its treatment, especially the dietary regulations. To this end the book is divided into thirteen sections, covering such subjects as General Health Rules, Medical Care, Food Principles and Calculated Food Tables with directions for planning, measuring and figuring the diet. There is also a section on Preventive Measures and one on Insulin.

The information in this book is complete and concise as far as it goes. While the calculated food tables are only approximate, they are sufficiently accurate

for the Dispensary patient who for various reasons cannot use an accurately weighed diet.

F. E. B.

Muscular Activity. By ARCHIBALD VIVIAN HILL, M.A. Sc.D., F.R.S., Professor of Physiology, University College, London. 115 pp. $2.75. (*The Williams & Wilkins Company, Baltimore, 1926.*)

The Herter lectures in past years have been quite uniformly of the highest quality and have generally been suitable for publications as monographs. The lectures given in 1924 by Professor Hill lend themselves admirably to this purpose. Here, in concise form, presented almost in narrative style, is an account of researches into the nature of the chemical and physical processes occurring in the contraction and recovery of muscle, researches which have won for their author the Nobel prize.

This monograph will be deeply appreciated, not only by physiologists but also by clinical workers who are interested in the effects of pathological processes on muscular activity, and in the adjustment of circulatory and respiratory mechanisms to the performance of work. In this connection, the fourth lecture, dealing with the recovery process after exercise, in man, will be of especial interest.

W. S. McC.

Schistosomiasis vel Bilharziasis. By C. G. KAY SHARP, M.D., with a foreword by J. B. Christopherson. 74 pp. $2.75. (*William Wood & Co., New York, 1925.*)

In this little book of 74 pages Dr. Sharp deals chiefly with vesical schistosomiasis and its causative agent Schistosoma haematobium. It is written for "the information of medical practitioners and educationists who have to deal with the problem of schistosomiasis." While it is chiefly a compilation from the original literature of the subject, it has as a background the experience of the author with the disease in the schools of Natal and includes a considerable amount of original material on the incidence, method of infection and clinical manifestations of the disease in South Africa.

In the part on etiology a discussion of the life cycle of the parasite is given and a description of the larval stages. There are also included descriptions and figures of the snail intermediate hosts gathered from papers on the mollusca which would otherwise be rather inaccessible to the medical man. The zoological aspects of the subject are not so well handled as the medical phases. The figures of the eggs of the three species of human schistosomes given on page 4 are so diagrammatic as to be of little value, and much better figures are available for the cercariae than those reproduced on page 17. In fact, the whole description of the cercariae is rather inadequately handled.

Following the etiology the disease is discussed under the headings of pathology, immunity, incidence, symptoms, diagnosis, prophylaxis, prognosis, and treatment. A well selected bibliography adds to the value of the work. In the sec-

tion on incidence the author adds to a review of the literature a number of interesting personal observations, especially on the incidence among school children in Natal and states that with a male school population in Durban of approximately 5,000 the incidence of the disease based on recorded cases is 2.4 per cent.

One of the most valuable parts of this little book is the section entitled "A general review of the treatment of schistosomiasis in different parts of the world in chronological sequence." The author concludes this section with the following statement:

"The foregoing review of the treatment can leave no doubt as to the specific action of the intravenous injection of tartar emetic in adults and emetine in children." He points out, however, that repeated examinations for a considerable time after treatment are necessary to establish cure.

On the whole, this book is a worthwhile contribution to the subject of schistosomiasis and will be a great help to those who need a condensed general account on vesical schistosomiasis.

W. W. C.

The George Blumer Edition of Billings-Forchheimer's Therapeusis of Internal Diseases. Six Volumes. Edited by George Blumer, M.A., M.D. $52.50. (*D. Appleton & Co., New York and London, 1925.*)

This is a new edition of a system that has long ago established its popularity with the practitioners of the art of healing, who must render first aid to the suffering while the scientist seeks out fundamental cures. The art must necessarily outrun the science of medicine, since the former is urged on directly by the cry for help. The physician is under the temptation to act, even when his judgment upon the efficacy of his therapeutic interference is held in the balance. One cannot therefore regard even a modern work on therapeutics with the same severely critical eye that one would bestow upon a discourse etiological or pathological. Even there one condones guesses; and in the healing art empiricism properly holds sway. Nowadays the reader has a right to expect, however, that the rationale underlying therapeutic procedure be frankly expounded, and that kindly groping be not masqueraded as securely founded indication. It is to the credit of the editor that, on the whole, this expectation has been met—there are numerous refreshing admissions of helplessness and ignorance. Where there are many contributors, as in this system, unevenness of treatment must be expected. No doubt some of the authors are blatant in their exposition of favorite therapies but their false pride serves all the more to emphasize the proper humility of the preponderating number. The most unfortunate contributions as in physical therapeutics and endocrinology are balanced by more critical discussions of the same theme by other writers in other sections. That the editor has not sought stricter coördination and regimentation of his staff is rather to be commended; for, in so doing, he stimulates the critical faculty of the student, new to the art; and focusses attention upon the fluent state of therapeutic science. By not exercising too severely his judicial and regulating function he teaches a desirable lesson in tolerance and open-mindedness.

The addenda to the previous edition are all timely and well considered, and the sections on Minor Surgery and the Special Sense Organs will be welcomed by many physicians who are in environments not favorable to strict medical specialization. By the experienced physician, most of the sections will be welcomed for review and reference. To the student and the young doctor a reading, even though hurriedly, of the work as a whole will afford a pleasing panorama not only of modern therapeutics, but also of disease pictures.

S. W.

"Allergy." By WILLIAM W. DUKE, M.D. 339 pp. $5.50. (*C. V. Mosby, Co., St. Louis, 1925.*)

This is a useful book for medical students and for physicians interested in Asthma, Hay-fever, Urticaria and allied manifestations.

In Part I, which consists of sixteen chapters, a comprehensive review of recent investigations, both experimental and clinical, is presented. The four chapters of Part II deal with the personal experience of the author with altered reaction in patients due to physical agents, such as light, heat, cold and mechanical irritants. A number of striking case histories are reported, with photographs of skin reactions to the various physical stimuli. Although this phase of the book is very interesting, the reviewer finds it difficult to accept it as allied to the subject matter in Part I. Certainly Pirquet did not intend his term "Allergy" to include such reactions as those which might occur in an individual who is exposed to cold, heat or light.

On the whole, the work fills the need of students who desire a summary of the hypersensitive states, and who have not sufficient time to read detailed investigations. The author supplies as an appendix an admirable and complete bibliography.

L. N. G.

Text-Book of Pharmaceutical Chemistry. By A. O. RENTLEY, PH.C., and J. E. DRIVER, M.Sc., A.I.C. 456 pp. $6.00. (*Oxford University Press, Lond., 1925.*)

This book contains an unusually complete discussion of the chemicals and galenical preparations of a chemical nature of the British Pharmacopoeia. Special emphasis is laid upon the tests for identity and purity of the official drugs, and interesting comparisons are made with Pharmacopoeias of other countries. The writers presuppose courses in inorganic and organic chemistry preliminary to the study of this book by pharmacy students. They, however, in an effort to make the book complete, review the fundamental principles of the science coincidently with the discussion of its pharmaceutical applications.

The chapter on the structure of alkaloidal molecules is especially complete and well written. However, it is unfortunate that the discussion of the alkaloidal evaluation of vegetable drugs, a subject of much importance, is not so complete as would be expected in a text of this kind. The latest developments in

the application of various phases of physical and colloidal chemistry, such as hydrogen-ion concentration and the preparation of metallic colloids, which play such an important rôle in modern pharmacy, are not discussed.

On the whole, the authors have given us a very valuable commentary upon the chemical constituents of the British Pharmacopoeia.

J. C. K., Jr.

The Early Diagnosis of the Acute Abdomen. By Zachary Cope. Third edition. 233 pp. $3.80. (*Humphrey Milford, Oxford University Press, London, 1925.*)

This book presents in a very convenient form a fairly complete discussion of acute conditions within the abdominal cavity. The author has drawn upon a large clinical experience; his conclusions on diagnosis and diagnostic signs are interesting and bear criticism well.

There is so much material that one feels some hesitancy in calling attention to two omissions. These are, first, Cullen's sign as a diagnostic feature of ruptured ectopic pregnancy, and second, the rupture of gastric or esophageal varices (splenogenous) as a cause of hematemesis.

This book will be found of great value to practitioners, students and teachers.

E. M. H., Jr.

Atlas of Diseases of the Skin. By Prof. G. Riehl (Vienna) and Prof. Leo V. Zumbusch (Munich). With Diagnostic and Therapeutic Notes by Dr. John B. Ludy, Philadelphia. Three vols. $25.00. (*P. Blakiston's Son & Co. Phila., 1925.*)

The three volumes contain altogether 84 plates. Each plate consists of two or three colored photographs which, in the majority of instances, reproduce very finely the skin affections which they represent. Only a few of the photographs are poor, most notably, perhaps, the one depicting freckles—a condition, fortunately, of little importance from a diagnostic standpoint. In a few others, the colors are a little too vivid or too pale, although otherwise the lesions are well represented. An occasional one is set off with variegated colors in dresses or cushions in the background which somewhat detract from the picture. Altogether, however, the volumes contain a very good and well diversified collection of skin affections, unusually well reproduced.

The diagnostic and therapeutic notes are, in general, concise, to the point and should offer an added attraction to the set. They could be improved, however, by a more uniform arrangement of the subheadings and presentation of the subject matter. Exception might be taken to certain items, such as "Senile changes may to a large extent be prevented by the frequent employment of emollient baths" and the too brief recommendation of trichloracetic acid for the removal of flat pigmented moles—a procedure which, if not thoroughly carried out, might result in dangerous malignant complications.

L. W. K.

The Mechanism and Graphic Registration of the Heart Beat. By Sir Thomas Lewis, M.D., F.R.S., F.R.C.P., D.Sc., C.B.E. 3rd edition. 529 pp. $12.00. (*Shaw & Sons, Ltd., London, Fetter Lane, Fleet Street, 1925.*)

This, the third edition of Sir Thomas Lewis's Mechanism and Graphic Registration of the Heart Beat, illustrates in an unusual way the great advances which have been made in our knowledge of the normal and pathological physiology of the cardiac mechanism. The first edition, appearing in 1910, comprised a volume of approximately 300 pages, which have been increased in the comparatively short period of 14 years to over 500 pages with the addition of an extraordinarily complete bibliography of over a thousand references.

This edition is conspicuous for the general revision which the text has received throughout and for the addition of several chapters of great value both to the student and to the more advanced worker in this field.

In particular we would call attention to the expansion of those sections devoted to a consideration of the normal electrocardiogram and the ventricular deflections, and to the chapters devoted to the nature of auricular flutter and fibrillation. These present the results of experimental work on circus movements that appeared from Sir Thomas Lewis' laboratory just too late to be included in the previous second edition.

The new chapters devoted to a discussion of the nature of extrasystoles and the parasystolic hypothesis of Kaufman and Rothberger are a notable addition and of the greatest value.

There is no other single text which includes so much or discusses in such authoritative and critical detail the fundamental problems involved in this fascinat ing and important field.

We can only emphasise the truth that no one seriously interested in the problems of cardiac physiology and the clinical manifestations of the cardiac irregularities can afford to be without, or at least to refer to, this volume. It contains all that is known at the present time of this most important subject presented in a way that leaves nothing to be desired, whether one agrees or not in every instance with the author's point of view. This volume is indeed a monument to the indefatigable enthusiasm and the brilliant critical interpretation and exposition of the writer.

E. P. C.

Clinical Features of Heart Disease. An Interpretation of the Mechanics of Diagnosis for Practitioners. By Leroy Crummer, M.D., Professor of Medicine, University of Nebraska. Introduction by Emmanuel Libman, M.D., Physician to Mount Sinai Hospital; Professor of Clinical Medicine, Columbia University, N. Y. 353 pp. $4.00. (*Paul B. Hoeber, Inc., New York, 1925.*)

This work differs from the majority of recent texts devoted to disease of the heart in several respects. It is essentially the medium of expression for the views of the author alone, containing little discussion of any other than the personal

point of view. There is but scant reference to the literature and no detailed analysis of the etiology of cardiac disease from the fundamental significance of the underlying morbid process, be it rheumatic disease, syphilis, bacterial infection or arteriosclerosis.

Throughout the author has emphasised the importance of the correct interpretation of the clinical signs and the necessity of an accurate evaluation of the changes that may be detected from day to day by careful clinical examination, the significance of which cannot be overstressed. Much emphasis is laid upon the interpretation of the character of the first sound at the apex and in adjacent regions—a most important desideratum. While it is true that the trained ear, under certain conditions, may be able to detect slight differences in what the author calls the "first sound right" from the "first sound left," any such sharp differentiation must always remain more or less difficult to the novice.

With the impression conveyed that the development of a tricuspid insufficiency in advanced mitral stenosis always leads to the striking changes as portrayed by the author, we are not wholly in agreement, though one may in some instances, it is true, see a diminution in the degree of cyanosis with the development of a tricuspid insufficiency. From the text one is led to believe that tricuspid insufficiency is a far less common development than is the generally accepted opinion.

As an exposition of the author's views upon cardiac disease and the importance of a careful thorough clinical study of every such case this volume is not without interest, though lacking the broader comprehensive perspective gained by a fuller consideration of the evolution of our present day knowledge and a more complete discussion of, or allusion to, the work of others in this subject.

In spite of the enthusiastic introduction, we have found the text somewhat disappointing.

E. P. C.

Gastric Function in Health and Disease. By JOHN A. RYLE. 152 pp. (*Oxford Medical Publications. Humphrey Milford, Oxford University.*)

In spite of the fundamental physiological studies which are on record dealing with the secretion and motility of the stomach, no subject in clinical medicine is in a greater state of confusion than that of the digestive disorders. The endless suggestions and hypotheses which are constantly brought forward to interpret clinical findings bear witness to this fact. Ryle's brief monograph on Gastric Function in Health and Disease is one of a number of recent books in which an attempt is made to stick to facts and to eliminate the fancy which has dominated this domain. The first section, on Gastric Function in Health, is a clean cut summary of what is known about the clinical physiology of the stomach, and is altogether commendable. In the second part, however, which deals with disease, the author has as usual split upon the rocks of attempting to define and classify that which is really not amenable to such treatment. As we see it, the best one can do is to study the reactions in the particular case—such reactions may or may not be compatible with a tentative diagnosis reached from history and physical

findings. For example, if a high gastric acidity with free secretion is found in a person who has symptoms often present in instances of gastric ulcer, the picture is fairly definite, but when an attempt is made to differentiate syndromes of habit dyspepsia, nervous dyspepsia, tobacco dyspepsia, anemic dyspepsia, irritative dyspepsia, and a host of others, with the idea that this is either feasible or useful, we must part company with the author's views.

A. L. B.

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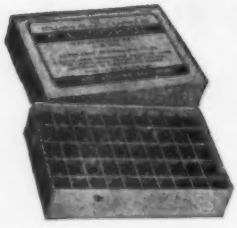

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